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MECHANISMS OF REGULATION OF THERMAL HOMEOSTASIS IN THE WORK PROCESS

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ABSTRACT. Thermal homeostasis involves numerous regulatory mechanisms. The thermoregulatory systems exhibit a duality, both topographic and functional, in that the centers which react to heat and to cold are not colocated. Specific mechanisms by which the body reacts to heat and cold are described.

The maintenance of thermal homeostasis of the human organism during work is /297* a particularly complex physiological process, owing on the one hand to the changes which tend to disrupt this equilibrium through increased production of heat during the exertion of effort, and on the other to the conditions of the environmental factors which influence and condition the level of thermal dispersion. Since it necessitates participation by nearly all the physiological apparatuses and systems of the organism, thermal homeostasis involves numerous and varied regulation mechanisms representing one of the most interesting chapters of applied physiology. This introduction of physiology into solution of current problems raised by the life and activity of the human organism represents one of the basic features of the new orientation in physiology. In this connection the work done by Academician Benetato and coworkers has made an important contribution to scientific elucidation of the mechanisms of thermal homeostasis of the organism in different types of activity, emphasizing the role and integrative importance of the neuroendocrine system.

As we know, the hypothalamic centers involved in thermoregulation are connected to the adjacent nuclei which control the glandular secretions, and to the zones which influence the tonicity of the vegetative nervous system; at the same time, these centers are subjected to influences from the level of the nervous paleocortical and neocortical structures through direct or intermediate relays (Kuhlenbeck, Sager, Slonim, Rioch).

The thermoregulatory systems exhibit a duality both topographic and functional, in the sense that the nuclear zones credited with the production of heat are located in the posterior hypothalamus, while those to which thermal dispersion is ascribed are situated in the anterior hypothalamus.

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* Numbers in the margin indicate pagination in the foreign text.

Despite the polymorphism of the physiological interrelationships involved in the generation and dissipation of heat, they are ultimately systematized in several modalities of reaction: chemical thermogenesis, tremulous thermogenesis, redistribution of the mass of blood, and secretory phenomena. The elaboration of heat is accomplished by biological processes involving primarily the production of heat in chemical and tremulous thermogenesis, but ones which /298 have relatively little effect on the cardiovascular system and basal secretory manifestations.

The thermolytic process, on the contrary, induces no substantial changes at the level of heat-generating cellular metabolism, it being accomplished mostly through redistribution of the circulating mass as a result of particular vasomotor reactions, and through major influence on the modalities of elimination of water from the organism.

The anterior and posterior hypothalamic centers enter into activity as a result of stimuli created by peripheral afferent influxes of heat or cold, and under the influence of the temperature of the blood which irrigates the diencephalic zone; these factors are of unequal importance, the importance depending on the temperature of the ambient medium. Thus at low temperatures the initiation of retrohypothalamic activity becomes decisive under the influence of the peripheral afferent vessels, while at high temperatures the major role is played by central sanguinary hyperthermy with anterohypothalamic repercussions. Through direct relays, or more often through intermediate effects, the thermoregulatory nervous structures affect both the topographically adjacent zones credited with neuroglandular activity and the physiology of the majority of the glands with internal secretion.

Viewed as a whole (Jacobs and Posner, 1964), biological hyperactivity of the posterior centers of the hypothalamus, heat generating, through intrahypothalamic relays entail overburdening of the axis T.R.F. (Thyriotropin Releasing Factor -- T.S.H. (Thyriotropin) -- Thyroxin and C.R.F. (Corticotropin Releasing Factor) -- ACTH-Hydrocortisone, this ultimately leading to thyroid and adrenocortical hypersecretion; at the same time, increased production is observed of the somatotropic hormone (S.T.H.) and of catecholamines, always with a high proportion of noradrenaline referred to the adrenaline values.

Despite the wide fluctuation in the proportion of the stimulus achieved at the hypophysial, thyroid, fasciculated corticoadrenal, and medulloadrenal levels and in the large variables determined by the intensity of the cold excitant and its coexistence with other environmental factors, the result of this complex of glandular changes is that at low temperatures the organism compensates for the most part by drawing on the lipide reserve.

In contrast to the foregoing, thermolytic mechanisms initiate endocrine and metabolic changes as a result of redistribution of the mass of blood, secretory reactions, and retrohypothalamic disconnection, the last-named being induced by suppression of peripheral afferent influxes of cold.

Owing to redistribution of the blood flow toward the periphery with resulting decrease in the renal blood flow, certain biological changes are

initiated the causal and chronological succession of which is juxtaglomerular cellular stimulation, hypersecretion of renin, overproduction of angiotonin I, and overproduction of angiotonin II (Blondheim, Kott, and coworkers, 1963).

Angiotonin II electively stimulates the glomerular zone of the cortico-suprarenals, this leading to secretory excess of aldosterone (Goldberg and Moore, 1963).

Intensification of anterohypothalamic activity as a result of suppression of inhibition from the posterior hypothalamus, the tendency toward hematic hyperthermis and peripheral afferent influxes of heat lead to hypersecretion of ADH, this also being fostered by hematic hypovolemia -- secondary to excessive sudation -- and by vagal hypertonicity effected by diffuse and elective blocking of specific acetylcholinesterase induced by bradyquinine (Gogata, Prunty, 1963). /299

At high ambient medium temperatures suppression of the hyperglycemic glycogenolytic activity of the catecholamines, and the hyperinsulinism due to increase in vagal tonicity, brings about a tendency toward hypoglycemia, which, against the background of a substantial decrease in glucocorticoid secretion encountered in a warm environment, under these conditions causes the organism to compensate for energy needs chiefly through the contribution made by hydrocarbonates.

Numerous recently conducted experiments have added to previous knowledge of the role of glandular secretions in thermoregulation. Thus in persons having undergone adrenalectomy the cooling rate is partly restored to normal by treatment with steroids. Persons suffering corticosuprarenal deficiency offer more difficult resistance to ambient temperatures. Opposite phenomena are encountered in persons with suprarenal-metabolic syndromes. Experiments conducted in this direction with animals having undergone adrenalectomy have shown that they die at a temperature of 22°, while normal animals survive at much lower temperatures. The survival of animals having undergone adrenalectomy and treated and exposed to a low temperature is a phenomenon independent of the rate of cooling at the same temperature, since small doses of hydrocorticoids incapable of restoring a normal rate of cooling nevertheless permit survival. Prolonged exposure to cold increases the ascorbic acid content of the corticosuprarenals, which content concomitantly increases progressively in the hypophysis as well, despite the fact that no progressive increases in the urinary ascorbic acid values have been detected. It should be pointed out, however, that among the endocrine glands the medullosuprarenal plays a decisive role in metabolic response to cold. Thus the ganglioplegics and adrenolytics impede increase in high consumption of oxygen in cold. Research by Sofer and Dorfmann has shown that the most important role in response to cold is played by noradrenaline and not by adrenaline. Substantial increases have also been observed in the elimination of 3-metoxy-4-hydroxymandelic acid (V.M.A.), the sulfo derivatives of normetanephrine, and the secondary sulfo derivatives of the transformation of metanephrine.

Research conducted in the Work Physiology Section of the Institute of Hygiene by Berdan, Pafnote, and coworkers has shown that in repeated exposure to heat the urinary elimination of 17-cetosteroids decreases 30 to 35% after 11 to 14 days.

According to Mallow the summary sequence of metabolic homeostatic phenomena in workers exposed to low temperatures comprises the following: (1) stimulation of the cutaneous thermoreceptors leads on the one hand to musculoskeletal tension with tremor and secondary thermogenesis, and on the other to hyperactivity in the posterior hypothalamic centers; (2) lowering the body temperature also causes hypertonicity of the centers in the posterior hypothalamus, hypertonicity which in turn causes adrenomedullary hypersecretion and sympathetic vegetative nervous stimulation, thyroid hypersecretion, and adrenocortical hypersecretion. The increase in adrenocortical and thyroid secretion causes deceleration of the rate of cooling of the body. Adrenomedullary hypersecretion, stimulation of the vegetative nervous system, thyroid hypersecretion, and the adrenocortical permissive effect result in increase in chemical thermogenesis at the level of /300
the liver, skeletal muscles, kidney, and gastrointestinal tract. We may also mention that it has recently been possible to demonstrate a decrease in the production of serontonin under low temperature conditions, the opposite phenomenon occuring in environments with high temperatures (Eikness and Hoel, 1963).

(Bibliography in the hands of the authors.)

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